DOCUMENT RESUME

ED 093 806 SP 008 170

AUTHOR	McCabe, Edward B., Ed.; Challop, Roger S., Ed.
TITLE	Highlights of the Midwest and Eastern Regional Conferences on Childhood Lead Poisoning; Chicago, Illinois, May 10-11, 1972 and Wilmington, Delaware, June 2-3, 1972.
INSTITUTION	American Academy of Pediatrics, Evanston, Ill.
SPONS AGENCY	Health Services and Mental Health Administration (DHEW), Rockville, Md. Bureau of Community Environmental Management.; Lead Industries Association, New York, N.Y.
PUB DATE	Apr 73
NOTE	32p.
EDRS PRICE	MF-$0.75 HC-$1.85 PLUS POSTAGE
DESCRIPTORS	Community Health; *Conferences; *Disease Control; *Government Role; Health Programs; *Lead Poisoning; Program Development
IDENTIFIERS	Chicago; Delaware; Illinois; Wilmington

ABSTRACT

Various aspects of lead poisoning and its control are the focus of this document which consists of edited speeches presented at childhood lead poisoning conferences in Wilmington, Delaware and Chicago, Illinois. Among the topics discussed are a) the role of Federal and State governments in lead poison control; b) the local government role, especially local health and housing departments; c) the role of the lead industry; d) treatment of lead poisoning for both ambulatory and hospitalized patients; e) legal and medical responsibility; and f) the development of lead poison control programs. (HMD)

ED 093806

Highlights of the Midwest and Eastern Regional Conferences on Childhood Lead Poisoning

Chicago, Illinois, May 10-11, 1972
Wilmington, Delaware, June 2-3, 1972

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Sponsored by the Illinois and Delaware chapters of
the American Academy of Pediatrics

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Financial support for these Conferences was provided by the Bureau of Community Environmental Management, Health Services and Mental Health Administration, DHEW. Financial support for this publication was provided by Lead Industries Association, Inc.

The Lead Industries Association, Inc. is pleased to publish these edited proceedings. However, it should be noted that some of the opinions and recommendations expressed herein are not universally accepted. Therefore, the statements must be regarded as the opinions of the authors, and do not necessarily reflect the views of the Lead Industries Association, Inc.

EDITORS' NOTE

Childhood lead poisoning, a many faceted problem of children everywhere who live in substandard housing, continues to receive attention from physicians, public health officials, housing experts, and politicians from around the country. Community child lead poisoning projects have been spurred recently by federal funds made available last year, after passage of legislation late in 1971. This increasing activity was the impetus for two regional conferences on childhood lead poisoning, held under the auspices of the American Academy of Pediatrics last spring in Chicago, Illinois, and Wilmington, Delaware.

The proceedings that follow are highlights from the two conferences. They represent edited versions of the entire recorded conference proceedings. Chicago, the site of the first conference, has had seven years of intense lead poisoning control activity, and thus, the presentation of technical reports, including much of the Chicago experience, dominated much of this meeting. By contrast, the second conference, in Wilmington, included many participants representing communities just beginning the struggle to organize lead poisoning eradication programs. Thus the flavor of the second conference differs from the first in that there is more emphasis on the aspect of community organization.

Because of our desire to make this document helpful to physicians, other health workers, and governmental officials, we have concentrated on presenting, in readable fashion, that information which emerged during the meeting and we expect will best further that goal. To those speakers whose talks were edited out, our apologies and thanks again for their participation.

A note of appreciation is due Mr. Marty Dale, Assistant Director, Department of Chapters of the American Academy of Pediatrics, for arranging the details of the conferences.

The participants of both conferences are listed at the end of the report.

April 1, 1973 Edward B. McCabe, M.D. Roger S. Challop, M.D.

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WELCOMING REMARKS – CHICAGO

Joseph R. Christian, M.D.
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In 1953 those of us who were involved in committee activities recognized lead poisoning as being a major problem in children, and wanted to do something about it, but no one would listen. Now we have everyone talking about it, and we are beginning to at last recruit people who are willing to listen. Our concern is that lead poisoning is a preventable disease, and in spite of this, we have not really been making any great accomplishments. In the past ten years, there probably has been more research done in the area of lead poisoning than at any other time. Those of you who have been interested in lead poisoning realize that this is a disease that was described very well in Roman times, and although people have begun trying to do something about it; thus far we have been unable to control our environment to the point that we are able to eradicate this scourge of childhood.

Childhood Lead Poisoning in Chicago – An Overview

Murray Brown, M.D.
Commissioner of Health, Chicago, Illinois

Until the 1960's, lead poisoning was recognized primarily as an occupational health problem, associated with the recovery of old lead from batteries. The inhalation of lead fumes from burning batteries used for heat in crowded living conditions was accepted as the major cause of lead poisoning in children. Then, in the early sixties, many clinicians and public health officials were beginning to recognize the consequences of ingested lead, but the significance of the problem was not appreciated because no data were available.

In 1963 in Chicago, lead poisoning was made a reportable disease, just as any communicable disease. By 1967, the first community screening program was under way. Twenty-eight thousand children were screened that year. At that time a blood lead concentration of 50 μg.% was considered the upper limits of normal for children. Approximately 650 of the 28,000 children who were screened had elevated blood leads identified that year, with evidence of symptomatic disease in a great number of these.

Since our lead poisoning program began in 1963, we have screened over 250,000 children. In recent years we have combined our efforts with other official agencies, such as the Chicago Building Departments, as well as unofficial groups such as the Chicago Committee on Urban Opportunity. Coordinated efforts have been aimed at identifying groups of children that should be brought under surveillance, educating community residents about the nature of childhood lead poisoning through the use of neighborhood health aids, and at deleading residences where children with elevated blood leads live.

Our latest effort has been the adoption of a city ordinance making it unlawful to use paint with a lead content of more than .06 per cent of the dried product on interior surfaces. This level coincides with the recommendations of the Bureau of Community Environmental Management and of the American Academy of Pediatrics. So legally we are making progress at the same time that we are making progress in the control of disease.

Our other area of interest in Chicago is in the research aspects of lead ingestion. In several of our clinical units child psychiatrists are attempting to understand the phenomenon of pica. What is the difference between children in the same family, living under similar conditions, who are known to exhibit pica behavior and those who don't? Can some approach be found that will identify factors that lead to pica in some children? Another area of interest involves the possible subtle neurological changes that occur following lead ingestion, but before "elevated" blood lead levels are attained. Recent evidence indicates that there are children who do poorly in school but who do not have lead poisoning by our present standards, but may be affected because they have ingested lead in sufficient quantity over a period of time.

With some data accumulated after six years of experience, we are beginning to understand the enemy; we are hopeful of overcoming him. We are far from satisfied with the results that we have at the moment, but don't let that discourage anyone. We have cut the death rate dramatically in children. What we have now, we must use and use vigorously.

Childhood Lead Poisoning – Conference Keynote

Agnes Latimer, M.D.
Assistant Director, Fantus Childrens Clinic
Cook County Hospital, Chicago, Illinois

I believe that the time is long overdue for us to commit ourselves to the task of planning for complete eradication of childhood lead poisoning.

Essential to the eradication of childhood lead poisoning is a more concerned attitude among health professionals toward the problem. One measure of this neutral attitude is the current controversy engaged in by health professionals over the description of the victim of lead poisoning. Some prefer to describe the child as having an increased body burden of lead rather than lead poisoning, although no one really knows what chronic, asymptomatic elevated lead exposure will do to developing brain and other body tissue.

The recent U. S. Public Health Service statement that "a blood lead level of 50-79 μg.% may not be associated with lead poisoning but a blood lead of 80 μg.% required immediate hospitalization and treatment with chelating agents" suggests some confusion in concept as well as terminology.

Our failure to eliminate lead poisoning hazards for children is rooted in the fact that it is primarily a disease of the urban poor and minority groups. It is very likely that if industrialists' children, doctors' children or legislators' children were afflicted with this illness, significant steps would have already been made toward its eradication.

All of us today who are attending this conference can be assumed to be concerned, in fact even shocked, by

the needless death and physical destruction which lead poisoning causes. Many and varied attempts have been employed in the past to understand and combat the lead poisoning problem, but most of the efforts have been short-lived and unsustained because they do not deal with the basic problem of the lack of housing for the urban poor. I believe what is needed is not more lead poisoning conferences, but a decision at this point and time, a commitment in fact, to eradicate lead poisoning in the next decade. This may seem like a very ambitious undertaking and, it is, but nothing short of total commitment to the eradication of lead poisoning will be sufficient. All other steps are at best pitiful charades enacted by people who know they will be ineffective.

Eradication of the disease is not impossible if one looks at the causes. Dr. Chisolm has described the causative factors resulting in epidemic lead poisoning as a triad consisting of a child, a parent and a place. The child is described as a toddler with exaggerated oral activities, but the age of greatest mortality with lead poisoning is between 12 and 24 months; this age period is when more than 50% of all toddlers of all classes will engage in mouthing and ingestion of non-food substances in their environment. The parent may be a mother with inadequate resources to cope with her family's needs. But the place is always the same – a neglected slum housing unit with lead flakes within easy reach of a child's grasp. We will always have toddlers with us, and we will probably always have parents with varying emotional and economic resources, but we must not continue to have the place.

Eradication of lead poisoning must begin with simultaneous immediate, intermediate and long term approaches.

Immediate steps such as screening of all children at risk in lead belts are available and are currently being carried out by all too few cities and states. Other immediate steps include widespread education about the hazards of peeling paint and plaster, with involvement of the community as an integral part of any organized program. The mothers of children with lead poisoning are a valuable source of information and education of other members of the community. Such educational approaches should be utilized in many settings and should strive to reach the family before the child is toddler age.

Intermediate steps include the effective handling of housing code violations by the creation of special courts to deal with these problems in order to shorten the interval between recognition of the violation and court enforcement. This court should be staffed by knowledgeable judges and lawyers to expeditiously deal with housing code violation. Other intermediate approaches could involve the training of teams of (unskilled) unemployed men to go into dwellings identified as having hazardous interior surfaces and removing the layers of paint and plaster in preparation for resurfacing and/or repainting. This must be a part of a concerted attack on lead poisoning and should be funded using tax incentives for the landlords.

Some long term approaches include the creation of a National Task Force on Lead Poisoning with the specific responsibility to develop a phased attack on the problem, including systematic elimination or repair of lead infested dwelling units and planned replacement by new low income housing units. In this connection, the current practice of allowing planned and funded low income housing units to be delayed by local, political and jurisdictional disputes is disgraceful.

Should you now say to me that this would be too expensive, let me remind you that conservative estimates of the cost, the treatment and institutionalization of children who sustain brain damage from lead poisoning through age 60 are in the neighborhood of over $100,000 per child, whereas the cost of repair of interior surfaces is in the neighborhood of $2,000 per house. We are already spending the money. It is a question of how we choose to spend it.

In summary, lead poisoning is a serious health problem afflicting over 100,000 children each year in the United States. We know the causes, we possess the resources and we can develop the methods to eradicate it. All we need is the commitment to do so.

The Role of the Community Health Center

John D. Madden, M.D.
Director, Woodlawn Community Health Center
Chicago, Illinois

The Woodlawn Child Health Center is an HEW supported children and youth project operated by the University of Chicago Department of Pediatrics as part of a city-wide program of child health services coordinated by the City of Chicago Board of Health and the State of Illinois, Department of Public Health. The goal of this program overall is the provision of comprehensive child health services to the Woodlawn community.

Woodlawn is a predominantly Black south side community which is underserved and which has the characteristics of such an underserved community: low income, high crime rates, high prematurity rates, high infant mortality rates, etc. Housing in Woodlawn is essentially all pre-World War II, predominantly multiple family dwellings and largely owned by

absentee landlords who fail to maintain it. As a result of this, lead poisoning has been endemic in Woodlawn.

In 1966 the Chicago Board of Health undertook a massive lead case finding effort employing blood lead determination as the screening test. This effort was so successful in finding children with elevated blood lead determinations that the traditional practice of hospitalization for diagnostic study and for treatment had to be modified. A decision was made to treat children with laboratory evidence of excessive body burden of lead but without clinical evidence of lead poisoning as outpatients instead of hospitalizing them.

The decision was made that the Woodlawn Child Health Center would not function as a primary screening facility, but rather would serve as a secondary referral center for the area. Any children who lived in Woodlawn who were detected to have a blood lead of 50 μg.% or more were to be referred to us for further evaluation and management.

In the early days of this program, all did not go well. We saw ourselves in a position of advocacy for the community and saw lead poisoning as the classic model of disease in the inner city – totally preventable and totally attributable to neglect of the needs of the urban poor. It was our position that lead poisoning was due to bad housing, purely and simply. Others tended to attribute lead poisoning to other causes such as maternal neglect. We were unable to accept this, having seen the anguish of mothers trying to care for children in dwellings that were unfit for habitation and in which landlords were loath to make repairs.

The initial days were stormy, since the referral system did not work well for some time. Now, however, we do receive regular referrals from the lead screening program. When we receive such a referral, we initiate contact with the patient's family. An appointment is made for a visit to the Child Health Center where the blood lead test is repeated, routine hematology is done, urinary coproporphyrin excretion is measured, abdominal x-ray and x-rays of the knees are performed, a history is taken, and physical examination is carried out.

The outcome of these procedures then determines the course of action. If the blood lead is marginally elevated and there is no supportive evidence such as anemia, 3 to 4+ coproporphyrinuria, basophilic stippling or x-ray evidence of recent lead ingestion or bone deposition of lead, then the family is counseled and advised as to the potential severity of the problem and the need for follow-up evaluation.

If there is a history of pica and especially if the housing is very bad, more careful follow-up is needed and efforts are made to assist the family in obtaining repairs or in relocating or in advising the family as to their rights as tenants and enabling them to get legal counsel.

When the blood lead is discovered to be between 60 and 80 μg.% and there is no supporting laboratory evidence, monthly monitoring of the blood lead is in order as well as attempts to correct the housing situation.

If there is a blood lead of 60-80 μg.% with supportive laboratory evidence for lead poisoning or if the blood lead is in excess of 80 μg.%, it is our belief that chelation therapy should be instituted with B.A.L. and E.D.T.A.

If the patient is asymptomatic and the blood lead is less than 120 μg.%, we believe it is safe to carry out chelation on an outpatient basis. If, however, there are symptoms suggestive of lead poisoning or if the blood lead is in excess of 120 μg.%, we are unwilling to treat the child outside the hospital.

In addition to the patients referred to us by the Board of Health, we carry out our own case finding within the population who use our Health Center.

Our principal screening tool has been the blood lead determination done by atomic absorption spectrometry. We have been impressed with this as a direct method of measuring excessive body burden of lead.

In 1970 we reported 85 children from our clinic with blood leads greater than 60 μg.%. In that report we attempted to correlate other laboratory studies with elevated blood lead. Ninety-four per cent of the children had hemoglobins of 10 gms.% or more. Only 19% had coproporphyrinuria greater than 2+ and only 11% showed basophilic stippling. Thirty-six per cent had "lead lines" on x-ray of long bones. This data seems to reaffirm our impression that the best screening tool to date is the blood lead determination.

Since May, 1969, we have treated 105 children with chelation therapy. These children have been treated principally as out-patients and were for the most part asymptomatic. We have found this to be a safe mode of therapy with no observable side effects of the therapy itself other than very occasional transient nausea with the institution of treatment.

We have observed a decreasing incidence of lead poisoning over the past few years. Forty children were chelated in 1969, twenty-six in 1970, thirty-five in 1971, and four to date in 1972. The 1971 figures are subject to skepticism because we found our laboratory at the University to have technical problems with the procedure during part of that year, a situation that has since been remedied.

It is difficult to attribute the decrease entirely to improved case finding and education. There has been a 30% drop in population in Woodlawn between the 1960

and 1970 census. We believe that this has occurred principally in the past few years and is directly related to the high incidence of fires in old buildings in East Woodlawn. We have had a decrease in population and a decrease in environmental source of lead. It is also true, however, that as Dr. Lin-Fu has recently stated, increased surveillance does lead to decreased incidence of lead poisoning.

What are the problems that the community health center engaged in a lead poisoning prevention program faces? They are many. The logistic problem of processing data and maintaining follow-up in a population that is very mobile is enormously difficult. The problem of interpretation of findings and decision-making as to who required therapy has plagued us but is becoming more clear-cut and its solution is aided considerably by the recent statement of the United States Public Health Service that provided specific (perhaps too specific) therapeutic guidelines for the management of lead poisoning.

The most discouraging problem is the housing situation. It is not a solution that is acceptable to have those at risk from lead trade that risk for the more immediate risk of fire. We must have more meaningful housing inspection and code enforcement. Landlords have to be made accountable and tenants' rights more adequately protected by the law. We must have new low income housing. Dr. Chisholm has reminded us many times that we must remove these children from risks of returning to inadequate housing. We support that as an ideal; that we cannot do it is too often the reality.

The problem that troubles the health provider in the field is the awareness that much of the problem and of the solution lies outside his capacity to bring about change through medical management. That is why many of us attempt to direct our energies, as citizen-physicians, toward the eradication of lead poisoning.

The Role of The Laboratory In The Prevention of Lead Poisoning

Elinor Berman, Ph.D.
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The literature is rich with descriptions of a variety of methods and techniques by which to judge exposure to lead and evaluate the degree of toxicity produced. The description of "lead lines" on the gums of individuals with markedly elevated blood leads and of basophilic stippling of the erythrocytes of such individuals are well known signs of undue exposure to lead, but are of limited value due to the infrequency of these findings. Stippled red blood cells have been shown to be present in about 25% of known cases of lead poisoning. The anemia must be quite severe. Furthermore, there are other clinical conditions which can cause an increase in stippled red cells.

In 1880 coproporphyrinuria was demonstrated in human cases of lead poisoning in Germany. However, urinary corprcprphyrin excretion is elevated by agents other than lead. Heavy metals like arsenic and cadmium can cause increased excretion; also various drug substances such as barbiturates, morphine, and even salicylates in some sensitive individuals can induce a coproporphrinuria. When employed as a screening procedure to indicate the need for further investigation, the urinary coproporphyrin determination, properly monitored, is acceptable, provided a more definitive test is used for confirmation.

Instrumentation and technical expertise evolving from modern technology are enabling us to determine elemental lead and other metallic contents in blood, urine, tissue and other materials with greater ease and specificity. The classical procedure used for lead determination back in the 1930's involves the formation of a color complex with a chelating agent, the most acceptable being dithizone. This procedure can be set up in a well-controlled, clinical chemistry laboratory and can be easily monitored.

Atomic absorption spectometry was introduced into the United States in 1962, and the first procedures for doing lead analysis with atomic absorption were published around 1964. The method has the advantage of being specific when one measures lead only. Since 1967, this method has been the most widely used, and in recent years analyses done with atomic absorption are the preferred method as far as the courts are concerned.

Private Practitioner, You've Missed The Diagnosis

Ronald Mack, M.D.
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University of Illinois School of Medicine
Chicago, Illinois

This morning I would like to discuss with you a heavy subject against which you can test your mettle. I am not mainly addressing my remarks to those physicians who care for children in the ghetto areas of large urban areas – be they private physicians in these areas or

those who work in public health clinics or those who work in county type hospitals serving the medically indigent. For it is almost inexcusable these days for such physicians not to think constantly of lead poisoning every time they seek a sick child – especially the preschool child. For the vast amount of recent communications in all media, both professional and popular, makes the omission of such a disease in the differential diagnosis of a sick child almost unforgivable. It is estimated that in the United States there are at least 250,000-400,000 children walking around with significantly elevated blood lead levels.

No, I would talk to those doctors who say they never saw a case or don't believe they will ever see one in their own practices because they mistakenly believe that their middle-class patients wouldn't allow themselves to contract such a disease. I submit that maybe some of you have some patients in your practice with subclinical lead poisoning and have probably had some in the past and have lost them and didn't know it. I believe you will certainly see some – if you look for them and think of the diagnosis. You will not see dozens of them in your practice, and that is one of the reasons why it is difficult to make the diagnosis; but there are reasons why I believe you will see lead poisoning cases in your own practices, and here are some of them:

(1) People are quite mobile and are becoming more so. According to the recent book, "Future Shock," they change dwellings frequently. In 1968, 36,600,000 Americans changed their place of residence. In each year since 1968, one out of every five Americans changed his address. What if a family with young children moves into an old house with old paint where active pre-school children never lived before?

(2) What if hospitals like the Cook County Hospital do close down? Where will these needy people go? They will possibly go to your hospital or clinic, and you had better prepare yourself for the problems of the underprivileged child, only one of which is lead poisoning.

(3) If the use of improperly glazed ceramics continues and increases, you will see more children as well as adults with this disease.

(4) If we don't do something about the ever-increasing air contamination due to lead from auto exhaust fumes, we will all see more cases, especially in children, since this air contamination can possibly increase the total body lead burden of children who have only small or marginal lead levels due to ingestion.

(5) It is true that the majority of cases at present involve inner city children from ghetto areas – but the results of the lead detection program as performed by the State of Illinois Department of Public Health identifies the problem as existing in many small communities. This type of program should ultimately be done in all states because it cannot be possible that lead poisoning in children only occurs in Chicago, New York, Baltimore, Cleveland, Boston, etc. Moreover, according to a recent article in *Pediatrics*[1] (Vol. 49, #4, April 1972, Dr. Michael E. Osband, Albert Einstein College of Medicine) another large high risk group of children that have not been properly studied are the children of migrant laborers.

The symptoms of chronic lead poisoning in childhood can best be described as nonspecific, protean, vague, ill defined, nebulous, insidious, not pathognomonic. This obviously makes diagnosis extremely difficult in the young child who cannot communicate his feeling and who does not enter your office or hospital emergency room wearing a sign, "I have lead poisoning." There are, however, a number of signs and symptoms that should at least arouse your suspicion enough to obtain proper lab tests to rule out this disease. That is what this is all about – to raise your index of suspicion.

The signs and symptoms of increased lead accumulation in the body chiefly involve three major organ systems in children – the gastrointestinal, the hematopoietic, and the central nervous system.

Usually the disease begins in a slow and subtle fashion with the child first exhibiting a diminution in appetite. He becomes progressively less playful and less alert. Associated with this is the gradual development of hyperirritability. Parents frequently state that the child has undergone personality changes, without being able to define these changes. If the mother is particularly observant, she may note that the child has undergone developmental regression; that he has lost his most recently acquired skills – he becomes clumsy and progresses to ataxia. These symptoms develop over a period of three to six weeks. During this phase, vomiting may occur intermittently and also brief self limited seizures occur. If lead ingestion continues, acute fulminant lead encephalopathy can occur.

It is fundamental to realize that we are primarily referring here to children 12 to 36 months of age – the age at which ingestion is high, and the age at which

[1]Osband, Michael, E., Tobin, J.R. Lead Paint Exposure in Migrant Labor Camps. Pediatrics, 49:604, 1972.

the child's curiosity is at its peak, particularly in exploring the environment with hands and mouth. Older children with this disease are usually retarded and have retained more infantile behavior patterns. Exceptions to these generalizations do occur – such as the older child who becomes lead intoxicated from drinking fruit juice from an improperly glazed earthenware vessel or among older children such as those in the Queensland, Australia, epidemic who amused themselves during the frequent rains by catching on their tongues the raindrops dripping from the colonial verandahs. These drops contained lead from the white lead paint on the roofs which was pulverized by the intense tropical heat and then washed down by the rains.

The gastrointestinal symptoms are those which are most likely to be noticed first by the parents and consist primarily of vomiting, vague abdominal pains, anorexia, and constitipation. These GI symptoms are very non-specific and are among the most common complaints in any group of pre-school children, as those of us in practice know so well. However, just for a minute or two when you hear this from a mother, think of plumbism no matter how remote the diagnosis seems.

Parents are often not of much help in your efforts to obtain definitive information. One survey of 300 children with confirmed lead poisoning showed that 76% had no presenting complaints, but when specific and detailed inquiry was made it was found that 58% had anorexia and 9% had vomiting. In another study of 22 children who had severe lead encephalopathy, it was found that 18 had been treated symptomatically for gastroenteritis for different periods of time before symptoms of central nervous system involvement became apparent.

I believe it is pertinent here to speak about pica, for the oral route is the major avenue of introducing lead into the body in children. There is an extremely high association of lead poisoning with pica – best described as a perverted appetite for dirt or other non food substances. As many as 50% of otherwise normal children, however, between one to three years of age eat non food substances, but some studies show that 70-90% of children with plumbism had a history of pica. Parents often deny this symptom, however. A careful questioning of the parents or their surrogates concerning pica must be made part of the routine questions asked in the development history of any pre-school child – sick or well.

The exact cause for pica is unknown. Many theories have been offered, some psychiatric, some instinctual, some physiological. For instance, it is believed by some that pica for lead containing substances is an attempt to replenish deficient body stores of iron. Although 90% of children with lead poisoning are iron-deficient, only one-third of the children with lead poisoning displaying pica cease the habit when given iron orally. I believe, as do many others, that excessive pica is probably a method of relieving anxiety by oral gratification caused ultimately by a disturbed mother-child relationship.

The hematologic findings will not be apparent to the parents, of course, and are less dramatic as initial complaints. Iron deficiency anemia is almost always present in young children with plumbism. This type of anemia is so common anyway in toddlers, especially in deprived children, that it is certainly nonspecific. Basophilic stippling of the red blood cells, as mentioned in all books and articles on lead poisoning, is apparently not that common in childhood as the spleen removes all the damaged cells rapidly. It does show well, however, on bone marrow examination of normoblasts. In the absence of an anemia due to an abnormal hemoglobinopathy or in such diseases as Cooley's anemia, the presence of basophilic stippling is thought by many to be pathognomonic of lead poisonings. The anemia is usually hypochromic and microcytic with a moderate reticulocytosis, polychromatophilia, poikilocytosis and an increased number of target cells. The anemia is brought about by the toxic effect of lead upon heme synthesis and by an increased hemolysis of circulating red blood cells.

The most serious manifestation of chronic or subacute childhood lead poisoning are those that result from brain involvement. The symptoms and signs vary tremendously and may range from drowsiness and lethargy to repeated grand mal seizures to coma and death. In some cases the first clues may be ataxia with repeated falling, also clumsiness and loss of coordination. Irritability, headache and insomnia occur. Persistent vomiting is usually an ominous sign of CNS involvement with cerebral edema. Acute encephalopathy is most common in children 15 to 30 months of age, while lead intoxication without encephalopathy in this age range and older can result in hyperirritability or aggressive behavior disturbances. The two to five-year-old with unrecognized plumbism may present with a convulsive disorder indistinguishable from idiopathic epilepsy or even a picture suggestive of a degenerative cerebral disease.

Acute encephalopathy is not usually seen after five years of age, but on the other hand the typical wrist drop, lead colic of the abdomen and lead line on the gums as seen in adults are not seen in young children. The neurological signs and symptoms are quite varied, quite nonspecific and often bizarre, and can mimic

several acute and chronic brain syndromes, especially tuberculous meningitis, any aseptic meningitis, brain tumor, poliomyelitis, hypertonic dehydration, traumatic subdural hematoma, and acute toxic encephalopathy from any cause.

There are a number of other peculiar ways that plumbism can present to the practicing physician, only to confuse and to delay proper treatment and recovery. One such presentation is lead myocarditis – not too many cases reported in children. Lead poisoning can also cause cardiac arrhythmias. Chronic renal syndromes can occur in children – not as many cases have been reported in the United States, however, as have been seen in other countries, especially Australia. Apparently it requires the ingestion of high concentrations of lead over a long period of time. Lead can also produce a Fanconi syndrome with hypophosphatemia, amino aciduria and glycosuria, much like the syndrome associated with outdated tetracyclines. Hyperuricemia and secondary gout have been reported in many cases of lead poisoning with chronic nephropathy. The nephropathy takes a long time to develop and the secondary gout comes even later.

Eighty to eighty-five per cent of cases of lead poisoning present themselves in the summer months although undoubtedly the lead has been ingested during the previous winter months. The reasons offered for this seasonal variation are not definite but suggest that the lead, being a divalent cation, acts like calcium and its absorption is enhanced by vitamin D, such as occurs with increased exposure to the ultraviolet rays of the sun. The increased heat and humidity in the summer can also enhance absorption and help to mobilize this heavy metal into the blood stream.

In summary, I would re-emphasize that all of us in the "front lines" of pediatrics think about plumbism during the course of our daily activities in our offices and in our clinics and hospitals. Learn to be very suspicious, especially in the summer months, especially in pre-school children who present with anemia and vomiting or lethargy or abdomen pain. Ask questions, especially during well-baby exams, concerning present dwelling site, recent moves, who cares for the child during the day. For instance, does the mother work and is the child cared for in a different home, an older home, possibly with chipped paint and falling plaster? Above all, ask specific questions concerning pica. Don't wait until you see a case of lead poisoning before you act. Practice preventive medicine. So much of what the office pediatrician does is preventative medicine anyway; why not include this? Finally, doctors, let us all get the lead out of the soft tissues of our buttocks and fight this man-made socio-cultural, technico-logical disease – or else, private practitioner, you have inexcusably missed the diagnosis.

Neurological Aspects of Lead Poisoning

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The neurological manifestations of lead poisoning are varied, and the major syndromes are: acute lead encephalitis; chronic lead encephalitis; cerebellar ataxia; convulsive disorder; behavior disorder; learning and perceptual disorders; mental retardation; and peripheral neuropathy. These are the eight major syndromes we have to consider.

Acute encephalopathy. Convulsions, delirium, coma and vomiting are the presenting symptoms and signs. The convulsions may be generalized or focal in pattern. Acute encephalopathy may develop after an initial period of irritability, anemia and anorexia, and it may be precipitated by acute infection or metabolic disturbance. In some cases, however, there are no premonitory symptoms or signs. Neurologic examination reveals papilledema, fixed dilated pupils, hyperreflexia or depressed deep tendon reflexes, Babinski signs, and often, nuchal rigidity. The skull x-ray shows spreading of the sutures, and the cerebrospinal fluid is under increased pressure with pleocytosis and elevated protein. This syndrome is most frequent in young infants during the summer months. If the acute encephalopathy is corrected, complete recovery is unusual and sequelae include hemiparesis, ataxia, mental retardation, convulsions, and optic atophy.

Chronic encephalopathy. After repeated nonfatal episodes of acute encephalopathy a chronic syndrome emerges resembling a progressive degenerative cerebral disorder, manifested by convulsions, ataxia, and mental deterioration.

Cerebellar ataxia. This may occur as the initial and main manifestation of central nervous system involvement but more frequently as a complication of acute or chronic encephalopathy.

Convulsive disorder. Convulsions may be generalized or focal in pattern. They may occur spontaneously or in association with fever and inter-current infections. They may be misdiagnosed as simple febrile seizures if they are short in duration and unaccompanied by changes in the cerebrospinal fluid.

Behavior disorders. These consist of irritability, frequent crying, inattention, impulsiveness, temper tantrums, and hyperactivity. Anemia, anorexia and loss of weight are frequently concomitant, but not invariable.

Learning disorders. Perceptual deficits include visual-motor incoordination and auditory imperception and the child with the learning disorder complicated by subtle neurologic abnormalities such as incoordination may be classified as having minimal brain dysfunction. Despite average intelligence, the child fails to achieve in school to the level of his potential. Improvements in behavior and learning have been reported after chelation therapy.

Mental retardation. Delays in development and regressions in intellect and behavior occur as neurologic sequelae of acute and chronic lead encephalopathy and have been reported in the absence of overt signs and symptoms of lead intoxication.

In one study of Perlstein and Attala in 1966,[1] among 58 children treated for asymptomatic lead poisoning, five, or 9%, were observed at follow-up examination to be mentally retarded. Admittedly, this study was retrospective, and one cannot be certain that mental retardation did not antedate lead poisoning. Pica, a common prelude to plumbism, may be a manifestation of emotional disorder or mental retardation, and the milder symptoms of lead poisoning are nonspecific and their significance often difficult to interpret.

Polyneuropathy. Wrist drop due to radical nerve involvement, a common manifestation of lead poisoning in adults, is uncommon in children. When neuropathy occurs during childhood, it usually affects the peroneal nerves and results in foot drop. Motor nerves and muscles subjected to the greatest use and fatigue are those chiefly affected. Diaphragmatic paralysis is a rare complication.

Differential Diagnosis

Acute lead encephalopathy must be distinguished from other forms of toxic encephalopathy, including arsenic and thallium poisoning and Reye's syndrome. Meningitis, particularly tuberculous meningitis, brain tumor and brain abscess should be excluded. Lead poisoning may occur at blood lead levels below 60 and even 50 μg per 100 ml, but some children with blood lead levels well beyond 100 μg per 100 ml appear well and asymptomatic. The finding of an elevated blood lead level alone should not be accepted as pathognomonic evidence of lead in the etiology of convulsions and coma.

Treatment of Encephalopathy

Convulsions should be controlled with phenobarbital and the cerebral edema treated with steroids and mannitol. Urine flow is established by appropriate fluid therapy, and the body temperature should be reduced to normal levels. Residual lead is removed from the bowel by enema and treatment with 2, 3-dimercaptopropanol (BAL) and EDTA by intramuscular injection is initiated and continued for five to seven days.

The use of surgical decompression is controversial and is usually reserved for patients in whom increased intracranial pressure persists beyond the initial five-day treatment period and in chronic encephalopathy.

Prognosis of Encephalopathy

A mortality of 20 per cent is to be expected and survivors sustain a variable amount of permanent nervous system injury. In one study of 425 children with lead poisoning, 39 per cent had some evidence of neurologic sequelae and among 59 children with lead encephalopathy, 82 per cent were left with handicaps.

Further efforts must be devoted to the prevention of lead poisoning, but the recognition of early symptoms and signs of intoxication and prompt therapy should reduce appreciably the number of fatalities and permanent neurologic sequelae.

Dose-Response Relationship for Lead

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I think it important that we put the problem of lead poisoning in its proper context, and the proper context really is dose-response relationship as applied to all toxins in our environment. The following remarks are based on information in a rather bulky document of some 500 pages, entitled "Air-Borne Lead in Perspective," recently published by the National Academy of Science. While the main information will be on children, I will refer to data obtained on adults where necessary.

If physicians are to deal effectively with the biologic effects of ubiquitous chemical agents such as lead, a working knowledge of the pertinent time-dose-response relationships is essential. Lead is currently considered a nonessential trace element and is detectable in minute amounts in food and water and in the blood, tissues, and excreta of virtually all healthy persons. Apparently, the biologic effects of the usual daily intake from uncontaminated food and water (approximately 0.2 to 0.3 mg lead/day in adults) is negligible, so that this small "dose" of lead has no known adverse effect on the health of people, but as the

chronic "dose" of lead increases, adverse responses of increasing severity become evident. While initial increments in "dose" (and absorption) produce obvious alterations in function and clinical symptoms of illness. Still higher "doses" will be fatal. Thus, a continuum of increasingly severe adverse responses are apparently associated with increasing doses of lead. The time factor adds yet another important dimension to the problem. A statement by the U. S. Public Health Service entitled "Medical Aspects of Childhood Lead Poisoning," sets forth rather detailed guidelines for the management of children with increased lead absorption, with emphasis on preservation of health and prevention of overt plumbism. An examination of some of the risk factors involved and of our scanty knowledge of the dose-response relationships to lead in children may help to place these rather detailed recommendations in perspective.

In the U. S. Public Health Service statement, great emphasis is placed on blood lead levels, which, in adults, reflect increases in the level of current and recent exposure to inorganic lead salts. In particular, the work of Kehoe in which human adult volunteers were fed supplemental amounts of lead (0.3, 1.0, 2.0 and 3.0 mg lead/day) has shown that blood lead levels increase in proportion to the dose and that the higher doses are associated with a more rapid rate of rise. While the relationship between blood lead levels and current exposure cannot be precisely defined, exponential increases in "dose" appear to be reflected by arithmetic increases in blood lead concentration.

Clinical observations in children are consistent with this observation. The "dose" of lead associated with the repetitive ingestion of a few lead-containing paint chips (or of putty) or lead-contaminated acidic juices may contain 10 to 500 times or more the quantity of lead found in normal diet – a truly enormous dose by comparison. Review of the basic data from previously published cases indicates that blood lead levels in children with pica for leaded paint may rise from the 40 to 60 μg lead range to well above the 100 μg lead range within a period of one to two months. How frequently this occurs is, of course, unknown, but clearly it can occur. Finally, the blood lead level does not appear to be in equilibrium with the total body lead burden, most of which is apparently rather tightly bound in bone; rather, the limited data available suggest that blood lead levels provide an index of the small but "mobile" pool of lead situated primarily in the soft tissues. It would further appear that this is the fraction of the total body lead burden responsible for the known acute toxic effects of lead.

Both in children and adults with lead poisoning, important interrelationships have been found between blood lead levels, "chelatable" lead, and quantitative daily outputs of aminolevulinic acid (ALA) and coproporphrin (UCP) in urine. Again, arithemtic increases in blood lead levels appear to be associated with exponential increases in these other parameters. Increased excretion of ALA and UCP indicates interference in biosynthesis of heme. The "chelatable" lead (i.e., response to a standardized parenteral dose of CaEDTA) correlates most closely with the daily output of ALA and UCP in urine.

Clinical risk factors may also be defined in terms of accurately determined blood lead levels. Epidemiologic surveys in "normal" adults indicate that mean blood lead levels are approximately 20 μg lead/100 ml whole blood (range 5 to 40 μg lead/100 ml whole blood) in populations without undue exposure to lead. Persons in close daily occupational contact with motor vehicular exhausts in confined spaces may exhibit blood lead levels in the 40 to 50 μg lead range, but rarely higher. Comparable data are not available in young children, since most of the published data are derived from populations in which pica, exposure to old housing and to air, dust and dirt-borne lead cannot be excluded.

Review of the files of previously reported children show that blood lead exceeded 100 μg in 136 of 139 cases of acute symptomatic lead poisoning. In 98 fatal cases of acute lead encephalopathy reported to the Baltimore City Health Department, the range of blood lead levels at the time of acute illness was 138 to 750 ($m = 330$) μg/100 ml blood. The range in 46 children considered asymptomatic was 55 to 300 μg lead/100 ml. While levels above 100 mg lead do not correlate well with the presence, absence, or severity of symptoms, virtually all such patients may be expected to show significant metabolic and functional abnormalities. Clearly, levels over 80 μg lead/100 ml whole blood in children signify a risk to health that is unacceptable. The biologic significance of sustained blood lead in the 50 to 80 μg lead/100 ml range is unclear: variations in metabolic responses are noted, but the usually mild and nonspecific nature of minimal symptoms precludes precise clinical diagnosis. Whether such children may sustain subtle but significant impairment of function, particularly in the nervous system, is not known. Nevertheless, such children clearly have a potentially hazardous increase in soft tissue lead and continued excessive intake at a high dose level can quickly cause severe illness. Prompt termination of their abnormal intake is the paramount consideration.

The recommendations of the U. S. Public Health Service statement for pediatric care vary according to

the estimate of current risk provided by blood lead levels. Levels less than 40 μg lead are assumed to indicate a negligible risk with normal daily dietary intake probably accounting for most of the intake. (These assumptions remain to be documented satisfactorily in young children.) Blood lead levels of more than 40 μg lead signify some additional source of intake and hence "undue exposure to lead," which may result from airborne or dust-and dirt-borne lead, especially in congested urban areas. If so, current estimates indicate that blood lead levels, at least in adults, are not likely to rise much above the 50 μg lead range. These sources do, however, serve to increase the background level upon which further intake must be superimposed. If, on the other hand, the child's undue exposure to lead results from pica for high dose environmental sources such as leaded paint, putty, lead in dirt, or the use of improperly lead-glazed earthenware pottery, a rapid rise in blood and tissue lead levels may be expected. Only serial determinations will indicate the trend and separate these two groups of children when the initial level lies in the 40 to 50 μg lead range.

Levels from 50 to 79 μg 100 ml of blood indicate an accelerating risk, an increasing probability of demonstrable metabolic impairment in heme synthesis, and the possibility of ill-defined symptoms of illness. Levels above 80 μg appear to present an unacceptable risk, particularly if long sustained. Chelation therapy is clearly indicated in this group, but would be of progressively decreasing benefit at lower levels of lead in the soft tissues. Because of the persistent and often clandestine nature of pica in some children, and the prevalence of deteriorated housing, serial testing of preschool age children in identified high risk areas would appear to be an essential part of regular health care. The trend of blood lead levels will help to determine the need for ancillary tests and indicated medical care in a given child.

The recommendations concerning ancillary tests are deemed by some as unrealistic in that they call for timed quantitative urine collection. This is a serious deficiency of the current "state of the art." Qualitative tests in random samples of urine for aminolevulinic acid or urinary coproporphyrin (ALA or UCP) are apparently inadequate in children, as they do not discriminate between normal and subclinical increased lead absorption. Since they become reliable indicators only as blood lead exceeds 80 to 100 μg lead, their usefulness is limited to children already at high risk. When reproducible simplified procedures adapted to capillary blood samples can be developed, the wider use of meaningful biochemical data, so often essential to good medical care, will be possible.

Perhaps the most serious limitation imposed upon good medical management of children (and adults) is the very limited availability to the general public of reliable lead analyses in blood and urine. A recent interlaboratory study revealed wide discrepancies in measurements from a number of laboratories. Reliable methods, quality control techniques, competent analysts, and continuous experience are essential if accurate and reproducible results are to be obtained in any trace metal analysis. The "U.S. Public Health Service method" has served in recent years as a suitable reference method for other dithizone techniques. Criteria for evaluating other and newer ones may be found in the analytical literature.

In conclusion, some may deem the U. S. Public Health Service statement too idealistic. It is, however, aimed at the preservation of health with guidelines based on the limited data currently available. Careful attention to dose-response relationships in studies in experimental animal systems and in clinical investigation may, in the future, help to define more closely some of the thresholds for the various significant adverse toxicological effects of excessive absorption of lead. At the biochemical level, the main adverse effects appear to be in the area of heme synthesis, cellular respiration, and membrane function. At the action level, it is clear that sustained pediatric follow-up during the pre-school years, coupled with better environmental control techniques, especially for the high-dose types of exposure, are essential to minimize this particular hazard to the health of children.

Ambulatory Treatment of Lead Poisoning

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During the first seven years of the 1960 decade, 1,200 cases of lead poisoning were reported in Chicago with 117 deaths. Community dismay over the high incidence of morbidity and fatalities, concentrated in only certain geographic areas of the city, prompted the Board of Health to initiate a broad lead screening program beginning in late 1966. In correlation with the case detection program, a specialized Lead Poisoning Clinic was established for evaluation and treatment of children found to have an elevated blood lead.

The screening program operates through eleven OEO-supported Urban Progress Centers from which a neighborhood canvass is conducted weekly. A Board of Health physician is in attendance at each Center once

a week for 3 to 4 hours to draw venous blood specimens. These are analyzed for lead content in the Board of Health Clinical Chemistry Laboratory. Blood lead is measured by atomic absorption spectroscopy. Hematocrit and hemoglobin electrophoresis are determined on each specimen submitted. Forty μg lead/100 ml whole blood was selected as the upper limit of normal.

The chief source of patients who attend the Lead Clinic is the screening program. About 10% are satellite cases or referrals from physicians, infant welfare stations or community clinics. About 1,800 new patients are examined in the Clinic yearly. Appointments are made by telephone or letter. If the appointment is not kept, a home visit is made by the UPC representatives or the district public health nurse.

Approximately 95% of children with lead values of 60 μg.% report to the Clinic. Those with higher lead values are seen within 24 hours or as soon as they can be located following completion of the laboratory tests.

In the Lead Clinic, history, physical examination and laboratory analyses of blood and urine are completed at the first visit whenever possible. A second venous sample is drawn, and x-rays are taken of the abdomen, wrist and knee.

If the initial lead value was 50 μg.%, the child is given a therapeutic dose of EDTA as a provocative test, and an aliquot of all urine collected in the succeeding 8 hours is analyzed for lead content. If the repeat blood lead value is 60 μg.%, injections are continued; if 40-59 μg.%, the patient may be started on oral penicillamine or we may choose to wait until the urine lead is reported. Treatment is usually withheld if blood lead is under 50 μg.% and the child is followed at 3-month intervals until the environmental hazard has been eliminated.

The patient is treated on an outpatient basis unless history or physical examination suggest impending severe encephalopathy. We routinely give a 5-day course of EDTA, 50 mg/kg with the addition of 15 drops of 1% procaine, as an intramuscular injection once daily. The site of injection is the anterolateral thigh. If much material is noted on KUB, Fleet enema is administered when the patient reaches home. Blood lead is repeated 4 to 7 days after the first course, and a second course given if blood lead was 80 μg initially. Urine is collected daily for 8 hours after each injection of EDTA to check the lead output.

Penicillamine is used if initial values were under 60 μg or after blood lead falls below 70 μg following one or more courses of EDTA. It is given orally, 125 mg two or three times daily, averaging 20 to 25 mg/kg. Medication is continued until lead falls below 50 μg.% for 2 consecutive months.

To date, the Clinic has seen 10,000 children, of whom 2,214 or 23% were treated, and the remainder observed until the lead hazard no longer existed in the home and blood level remained below 50 μg.% on two successive tests. A history of paint ingestion is next in importance to elevation of blood lead in making a diagnosis of lead poisoning. Eighty-one per cent of our patients were observed to eat peeling paint from walls and woodwork, or broken plaster from the walls. Metaphyseal lines wider than 1 mm were also noted in 80%. Although credence is fairly definite at 2 mm, the 1 mm line was regarded as a worthwhile cue. Opaque material in the gut was present in 40%; screws, staples, coins, beads and open safety pins were unusual despite frequent assertions that the child puts everything in his mouth.

Symptoms were present in 8%, from loss of appetite to vomiting and drowsiness. A few children with convulsions were picked up in the initial telephone contact and promptly hospitalized at Cook County Children's Hospital.

About 10% continued to eat lead-containing material for varying periods, as demonstrated by x-ray of the abdomen, by a rise in blood lead or by a failure to drop as anticipated. This recurrence rate is about half that recorded by other observers.

Most of the subjects who presented with values of 60 μg.% or more were treated; however, there was a great deal of selection of those in the 40 and 50 μg range. There were 465 children over 80 μg.% and close to 400 in the 70's. This is a terrifying potential [illegible] lead deaths, and one that makes us question the validity of only 117 reported deaths in the period from 1960 through 1966.

Conclusion. During 1967 and '68, there were 25 deaths attributed to lead poisoning, but in the three years following, there were only 8. The ambulatory Clinic has made a significant contribution to lowering the death rate and preventing hundreds of cases of encephalopathy, with its almost inevitable sequelae of brain damage and neuromuscular defects. This has been accomplished by bringing a sense of urgency to the early recognition and prompt detoxification of children with this insidious, catastrophic disease.

Treatment of the Hospitalized Patient

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The prime indication for hospitalizing the child with lead poisoning is encephalopathy. Signs of encephalopathy include changes in personality,

ataxia, vomiting, convulsions and somnolence. Children of the age group at risk who manifest any of these symptoms and are suspected of lead poisoning should be admitted immediately to the hospital for study and treatment.

Asymptomatic cases in which screening tests reveal very high blood levels should also be hospitalized. If the levels are above 100 μg./100 ml. hospital treatment is indicated in our opinion because of the great risk of sudden deterioration.

A third indication for hospitalization is inability to remove a child with a high blood lead level from continued exposure to lead ingestion while he remains at home. Under these circumstances, we believe the patient should be hospitalized and treated for his lead poisoning while measures are taken to insure that he will return to a home in which the risk of lead ingestion is not present.

Immediately after admission of a child suspected of having lead poisoning to the hospital, it is essential that confirmation of the diagnosis be obtained. A careful history is essential with emphasis on not only the symptomatology but also on whether pica is present. Careful initial physical examination is of importance particularly for neurological abnormalities. Laboratory examinations should be done at once and should include complete blood count and hematocrit, urinalysis, test of the urine for coproporphyrin, and x-rays of the abdomen, skull and long bones. The most important test, of course, is the blood lead level. With the development of atomic absorption spectrophotometry the test for blood lead has become rapid and reliable.

Early involvement of the Department of Social Service is also important. It is essential that social service workers begin their work immediately in order to arrange for new housing. It is also essential that other children in the family be brought to the hospital for examination and that notification of public health authorities be made if the diagnosis is positive.

Therapy for lead poisoning in the hospital should be started as soon as the diagnosis is established. The immediate objective is to obtain adequate urine flow. If the child has been vomiting and is dehydrated, urinary output may be inadequate to excrete the lead which will be chelated. Therapy can be started with 10% glucose administered intravenously at 10-20 ml/kg over a 2-hour period. After this, basal requirements are met in a dose of 350 ml/m² plus urinary output. It is extremely important to avoid excessive fluid input, especially that which contains excessive amounts of sodium chloride, since such fluids may cause development of cerebral edema.

Following the establishment of urinary flow, therapy is started with the chelating agents. We employ both dimercaprol (BAL) and calcium disodium versenate (or edathamil calcium disodium) EDTA. Treatment is started with BAL, which is given in a dose of 4 mg/kg. After this initial dose, BAL is repeated every 4 hours in a dose of 4 mg/kg for a period of 5 days. Starting with the second dose of BAL calcium EDTA is given intramuscularly at other sites in a dose of 12½ mg/kg every 4 hours. Small amounts of procaine are added to the EDTA to reduce the local pain. Although EDTA is a more effective chelating agent for lead than BAL, if one treats with EDTA alone, on occasion there may be during the first day of treatment a sudden deterioration of the patient with rapidly progressive encephalopathy. It has been suggested that treatment with EDTA may mobilize lead which is not adequately bound. As a result, more of the lead can enter the brain and cause additional symptoms. This can ordinarily be avoided if therapy is started with BAL.

Enemas should also be avoided. At one time therapy with chelating agents was delayed until the bowel was emptied, since it was felt that there might be increased absorption of lead from the bowel after chelation therapy. We no longer believe this to be true. Enemas can result in excessive hydration of the patient and can encourage development of cerebral edema. Treatment may be delayed for many hours while waiting for the enemas. Lumbar puncture should also be avoided whenever possible. In the presence of increased cranial pressure, lumbar punctures are not without risk. If there is a serious question of diagnosis between lead encephalopathy and pyogenic meningitis, then a careful lumbar puncture with removal of minimum amounts of cerebral spinal fluid may be necessary. This is seldom the case in our experience. Neurosurgical procedures such as surgical decompression should be avoided in the treatment of lead poisoning. Newer methods of therapy have made these procedures unnecessary. Both morbidity and mortality are increased. In the presence of severe cerebral edema intravenous mannitol can be employed in a dose of 1-2 mg/kg as a 20% solution. In cases with marked elevation of temperature, a water mattress can be used to reduce the fever. If respiratory distress or respiratory arrest occurs, assisted respiration may be necessary.

After the five-day course of chelating agents for lead poisoning it is essential to determine whether a second course is required. Evaluation of symptoms and serial blood lead levels are helpful in this respect. If blood lead levels remain high, a second course of de-leading again is employed for a period of 5 days.

After chelation treatment has been completed, it is essential to evaluate the child carefully for evidence of gross neurologic damage. The child's parents should be informed of the importance of avoiding new ingestion

of lead. It is essential that new housing be arranged for the family. The difficulties involved may be great, but new housing is essential to avoid recurrent lead poisoning.

The child should be followed carefully in the outpatient department in order to determine any neurological residual of the lead poisoning. Residual effects include gross neurologic impairment, loss of intelligence, behavior disorders and convulsive seizures. If a convulsive disorder develops, proper anti-convulsive medication should be given. If mental retardation is present, then proper school placement should be arranged at the proper time.

The results of our inpatient therapy of lead poisoning at Cook County Hospital are of interest. From 1959 through 1970 at Cook County Hospital we had 1,750 inpatient cases of lead poisoning. This included 233 patients with encephalitis. There have been 72 deaths (31% of the cases with encephalitis, 4% of the total cases).

Data from recent years indicate that while the number of cases of lead poisoning remains high, the number of cases with encephalitis has dropped off markedly. I believe that this is the result of better education, better screening and earlier diagnosis. It will be noted that from 1959 to 1963 there were a considerable number of deaths each year. This has dropped off rapidly, so that last year we only had one death from lead poisoning. We feel that the reduction in deaths reflects fewer cases with encephalitis. We feel it also reflects improvement in therapy. We feel this has resulted not only in the reduction in mortality but also in reduction of prominent after-effects from lead poisoning.

In a true sense, however, even "successful" treatment of lead poisoning and lead encephalopathy in children still represents failure. Our basic objective must be the prevention of this unnecessary but dangerous disease. Although physicians have done much with regard to the diagnosis and therapy of childhood lead poisoning, their major contribution will have been to call to the attention of the public the presence of this silent menace in our community. Prevention – not inpatient or outpatient treatment – must be our primary objective.

Industry's Role in Preventing Lead Poisoning

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The Lead Industries Association, Inc. is a non-profit organization of representatives of lead mining, smelting, refining and fabricating companies throughout the non-communist bloc of nations. Among other things, its purposes are to disseminate accurate information regarding lead products and how they are properly used, and to promote the service of lead industries to the community at large.

As a continuing part of our interest in the community, we have for many years, both alone and in cooperation with governmental, professional, industrial and academic groups, worked towards the elimination of the tragic problem of lead poisoning in children. Since the 1920's the lead industry has supported research on the metabolism of lead and the diagnosis and treatment of lead poisoning in an effort to bring about better understanding of the problems of plumbism. Such studies have been carried out at numerous educational institutions and private laboratories throughout the world. Information developed under our sponsorship has been distributed in medical and public health journals, and at various lead symposia conducted by LIA, both alone and in cooperation with organizations such as the American Medical Association and the American Industrial Hygiene Association.

As we know, progress is being made in many cities to bring the problem of pediatric lead poisoning under control and to reduce or eliminate deaths or serious disabilities resulting from lead paint ingestion. However, lead problems are still occurring despite many years of effort, and will continue to occur in children who live in old and poorly maintained homes that are not yet free of old leaded paint.

"Lead Poisoning in Children," published by the Children's Bureau of the U. S. Department of Health, Education and Welfare, states that lead poisoning was not found among children in newly constructed housing projects in New York City, even when these were in districts which had a high incidence of childhood lead intoxication. This indicates that lead poisoning is unquestionably linked to old dilapidated housing. This observation was further confirmed by a well-known study in Cleveland, where it was reported that of 801 young children in old housing, 38 children or 4.7 per cent had plumbism, while not one case of plumbism was found among 105 children who lived in a new housing project.

The Lead Industries Association has supported and encouraged the efforts of the Federal Government, states, localities and community action groups in the formulation and enactment of effective programs to eliminate the source of childhood lead poisoning. We have developed a proposed municipal ordinance, based on the Philadelphia law and its effectiveness, which is

available to communities upon request. The Lead Industries Association will continue to cooperate with all groups sincerely working toward our mutual goal of preventing lead poisoning in children.

Defining the Extent of Lead Poisoning – The Role of the State Health Department

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Although lead poisoning or plumbism has existed since antiquity, it has only recently attracted considerable attention as a problem of public health significance. Classically, two distinct categories of lead poisoning have existed: occupational lead poisoning and pediatric lead poisoning. The former has generally been associated with workers employed in specific types of manufacturing processes, while the latter has been recognized primarily as a problem of larger inner city urban centers. There, a "marked concentration of lead poisoning cases occur in slum areas-the so-called lead belts." In these areas, as in communities elsewhere, lead-based paint was commonly used on interior surfaces until the 1940's when they were largely replaced by titanium-based paints.

Conspicuous by its absence in the literature, however, is discussion of the basic issue of whether elevated blood lead levels and lead poisoning cases might occur in areas outside of recognized urban lead belts and may, in fact, be endemic to any areas where lead based paints and housing in deteriorated conditions occur. Seeking an answer to this question and also to initiate a direct service activity, the Illinois Department of Public Health undertook the Nation's first statewide program to assess the extent of elevated blood lead levels of children in communities of intermediate population.

There are about a million children in Illinois between the ages of 1 and 6, six months and six years; 333,000+ reside in the metropolitan Chicago area, and that means there are 666,000+ downstate. The remainder of the state's population is divided nearly evenly between communities of less than 10,000 people, including rural areas, and communities between 10,000 and 150,000 population. So the 1971 program of the Illinois Department of Public Health addressed itself to the latter group of Illinois communities of intermediate population.

A group of potential target cities from the 149 in Illinois with populations between 10,000 and 150,000 was identified. Selection of cities was made on the basis of geographic location, racial composition, population density, available medical facilities, known civic and community action groups and the presence of a functioning county or city health department.

Blood specimens were collected with sterile, 12 cc disposable syringes fitted with 1" disposable needles. Aliquots of blood were transferred into lead-free heparinized vacutainer tubes for lead and hemoglobin determinations.

All tubes were labeled with the patient's name, date and city, and all this was done by people from the community. Specimens were refrigerated at the test site until each city's program was completed (never more than 48 hours), and were then transported by special ambulance to the Hektoen Institute in Chicago.

The 5.0 ml. of heparinized whole blood for the lead determination was hemolyzed in distilled water, chelated with sodium diethyl dithiocarbonate at a pH of 6.5, extracted with methyl-iso-butyl ketone and analyzed with an atomic absorption spectrophotometer, using a single slot burner and a wave length of 2170 A°. Pooled samples were run daily along with the program specimens. The standard deviation of the results of these pooled specimens was 3.0 μg.%.

The guidelines of the U. S. Public Health Service were used to determine the significance of the laboratory results. Therefore, a blood level value of 40 μg.% or greater was considered evidence of undue absorption of lead, and a blood lead value of 80 μg.% or greater was considered an unquivocal case of lead poisoning.

To give the results: 6,151 children were screened in 14 cities. Overall, in the 14 cities visited, 6.14% of the total population 1 to 6 years of age was tested. Over 5% of the children in the "at risk" age group were tested in 10 of the 14 communities, and over 10% of children in the "at risk" age group were screened in 4 of these communities.

The volunteer effort in each community was successful in attracting a sufficient number of children to attain the first objective of the program – that of 250 children per community. We averaged about 440 children per city.

The mean blood lead levels ranged from a low of 19.8 μg.% in Rockford to a high of 32.9% in Peoria, with the mean blood lead level of the total population screened determined to be 25.5 μg.%.

The number of children with blood lead values 40 μg.% or greater ranged from 13 in Robbins to 202 in Springfield. The percentage of children with blood lead values 40 μg.% or greater ranged from 9.6% in Rockford to 31.3% in Peoria. For all cities screened, the number and per cent of children with elevated blood

lead levels (40 μg.% or greater) were found to be 1,147, or 18.6% of all those we screened.

Fifty-one children (0.8%) were found to have blood lead values of 80 μg.% or greater.

It is interesting that the City of Aurora differed markedly from the other thirteen cities in the percentage of children with blood lead levels in excess of 80 μg.%. About 4% of those children screened were found to have values equal to or greater than 80, whereas none of the others even came close to that.

Eighty-one and four-tenths per cent of the children screened were found to have blood lead values below the 40 μg.% cutoff point. Hence, 18.6% of the children screened demonstrated blood lead values indicative of undue absorption of lead, while 0.8% were classified as unequivocal cases of lead poisoning, by the U. S. Public Health Service guidelines.

It is interesting to note that if the distribution of blood lead values is lumped by 20 μg.% groupings, nearly one-half of the children (44.5%) demonstrated blood lead values in the 20-39 μg.% range, while only slightly more than a third (36.9%) showed values less than 20 μg.%.

In summary, it should be emphasized that the Surgeon General has estimated that upwards of 400,000 children across the nation may possibly have elevated blood lead levels. If the Surgeon General's estimate was based upon the previously accepted hypothesis that elevated blood lead levels are confined to major metropolitan areas, then it would seem that this estimation of the extent of the problem must be revised upward to include the large numbers of children outside of major metropolitan areas who are also affected. For, as this study has shown, elevated pediatric blood lead levels do exist in considerable numbers in communities of intermediate population.

Nursing's Role in Lead Poisoning Prevention

Jane Reed, R.N.
Nurse-Consultant, Bureau of Community Environmental Management, HEW
Cincinnati, Ohio

Nurses' skills in casefinding, education, supervision, coordination, and patient advocacy provide a powerful resource for communities, physicians, and health care facilities committed to eradication of this completely preventable disease.

Specific activities of community health nurses may vary depending on their work setting and level of involvement. The "front lines," namely, public health home care, health centers, pediatric outpatient departments of hospitals, and well child clinics should be the areas of greatest concentration of efforts.

Casefinding directed toward detection of those presymptomatic children with increased lead absorption can be best accomplished through pursuading parents to bring their children for testing. Door-to-door canvassing and mobile units may also be employed in the screening.

Community health nurses' roles, again depending on the situation, may be direct or supervisory.

In the direct role, nurses may identify high risk children using the child's age, housing conditions and habits as criteria.

Age – between 1 and 6 years, especially those in the 1 to 3 year range;
housing – old with flaking paint and plaster inside or out; and
habits – putting "things" in his mouth other than food. All children go through this stage, and lead problems arise when they continue to "try out" the paint and plaster.

The importance of maintaining and *projecting* a non-judgmental attitude on the part of the professionals and all those working with parents of lead poisoned children should be stressed. Lead poisoning is not caused by uncaring or malicious parents. It is the result of unawareness.

In some situations it may be better to inquire about the presence of flaking paint or plaster than to ask about pica outright. Some surveys have shown that the correlation between flaking paint and plaster and elevated blood lead levels is better than between pica and elevated blood lead levels. The pica question may also be interpreted by an anxious parent as a question of their adequacy.

Screening may be done "on the spot" or at nearby centers. If the child or children are found to have evidence of increased lead absorption, removal of the lead source is necessary to stop exposure. Sources may be many and varied – the child's residence, a neighbor's house, the grandmother's house, the play area dirt, etc., a listing of the places where the child spends his waking hours will be helpful in determining his lead sources.

In most situations, assistance will be available from sanitarians or housing inspectors to test paint and plaster and part of the follow-up is making sure this has been done.

Their activities should include discussing the results of tests and answering parents' questions when indicated, explaining follow-up and assisting the parents in finding resources for dealing with specific problems, such as housing, dietary counseling and anticipatory guidance may begin at this stage also.

Many programs will encourage and employ community residents to do casefinding, education, specimen collection, and follow-up. Nurses will be responsible for training, supervising and supporting these most valuable team members.

Open, easy communications are important for smooth functioning and optimum patient care. Each team member must understand the importance of his job and program goals, the ultimate goals being eradication of childhood lead poisoning.

Lead Poisoning and the Local Health Department

Patricia Wendling
Lead Poisoning Control Service, St. Louis Division of Health
St. Louis, Missouri

A municipal health department's role in the prevention of childhood lead poisoning is necessarily limited by three factors: (1) its traditional pattern of functioning in public health activities; (2) the politics of the particular issue; and (3) the insufficient appropriation of resources to the particular cause. Consequently, most local health departments seem to have accepted Jane Lin Fu's concept of prevention – prevention "through early detection and termination of undue exposure." In accordance with this concept, they have accepted roles as the detector of and resolver for elevated lead levels and actual cases of childhood lead poisoning. Certainly, those who are involved in public health programs committed to the elimination of this disease realize the "last resort" nature of this type of prevention and experience the daily frustrations which are built into preventing the further development of a problem rather than preventing its origination. Most local health departments' lead poisoning control programs have been built around this concept of prevention and its implied roles.

In the area of providing health-related services, many common problems are encountered by the majority of health departments; the absence of Federal funds for the support of all-out comprehensive educational campaigns against childhood lead poisoning, health center transportation systems, and a reliable micro-blood-lead technique severely limit the detection and resolution capabilities. The high mobility rate of the families involved also make close follow-up difficult in most cases. In some instances, the families cannot be reached at all.

Childhood lead poisoning is a man-made public health problem, and the real answers to its prevention in absolute terms are not unknown. The fact, however, that it has reached its programmatic level in a municipal health department demonstrates that the real answers are presently either "unfeasible" or "unacceptable" as one prominent urbanologist has suggested of most solutions related to our urban problems. For instance, this disease could be prevented by providing adequate housing for all low income families or by completely ridding dwelling units of lead-base substances, but both are economically "unfeasible" and "unacceptable."

The local health department, under the present circumstances, fills a necessary role in the vacuum of real childhood lead poisoning prevention. Its preventive role, however, must be well-defined to avoid misleading the community, to enable the development of realistic program objectives, and to bring out the need for real and necessary institutional solutions. We are seeing some hopeful signs in this area, such as housing and lead-paint legislative activities at all levels of government, recent appropriations to low income housing and lead-base paint poisoning prevention programs, and increasing community awareness to the problem; however, in view of the slow pace and slight magnitude of these developments, the need for the "last resort" role played by local health departments in the prevention of childhood lead poisoning will be around for a long time.

Role of the Housing Department

William Burke
City of Chicago Building Department

For an effective lead poisoning prevention program, close cooperation between a number of both public and private agencies is necessary. The Board of Health and the Building or Housing Department need to work jointly to provide the various services to the community at risk. Since the problem of lead poisoning is as much an environmental problem as a medical one, this dual responsibility is mandatory.

Building Department inspectors should be cognizant of the fact that a paint chip the size of a dime, ingested daily for two to three months, is more than enough to cause lead poisoning. The need for taking samples is being greatly reduced by the use of x-ray fluorescence analyzers. These portable, non-destructive testing units give an immediate reading in milligrams per square centimeter. X-ray fluorescence analyzers have been in use for screening for over a year in many cities with good results.

The system for dwelling unit inspection will depend on the resources available in the community. A comprehensive data collection mechanism should be developed, aimed at gathering relevant information about the specific dwelling unit and its occupants. The location of hazardous lead paint should be detailed, and if necessary, samples should be obtained for analysis and possible use for litigation purposes.

Details for deleading should be worked out. Voluntary compliance is probably ideal, but often additional steps to assure prompt deleading become necessary. A number of municipalities have created ordinances aimed at forcing landlords to have dwelling units in which small children live made safe. Some communities have developed deleading teams, employing inner city youth for the work, utilizing model city funds.

There are a variety of methods used to delead a dwelling unit. Basically, the hazardous painted surfaces need to be covered or removed. The cost of deleading varies, but most place the figure at about $400 per room.

The Role of the Federal Government

Robert J. Laur, Ph.D.
Deputy Administrator
U.S. Health Services and Mental Health Administration

If we really wish to prevent lead poisoning we have to recognize it also as a housing problem. It is a complex problem that requires a partnership of effort from all levels of government and a great variety of organizations in the private sector as well. The Federal Government tried to come up with a strategy which at least makes that partnership possible.

The national approach maintained that a comprehensive strategy of dealing with lead poisoning is needed, and not only are a lot of partners involved, but screening of children to detect those at risk, bringing those who are exposed to the hazard or have developed symptoms, into treatment, finding the housing units from which they come and taking remedial action in those units, are all parts of the strategy. All pieces must be there or the strategy is worthless.

It has been recognized also that the Federal Government had better get itself together if it is going to try to cope with the problem of lead-based paint poisoning. It has been proposed that an interdepartmental committee be formed of several governmental agencies that have an interest in this matter. For example, Housing and Urban Development is critical if the Government is going to have an effective role to play in doing something about housing. The Environmental Protection Agency clearly has a role here, as does HEW, and the Council on Environmental Quality. There may be others. Attempts are being made to bring all those departments together in a coordinating committee for the purpose of lead-based paint poisoning control.

Within HEW, such an intradepartmental committee has already been formed. The Food and Drug Administration, the National Institutes of Health, the Health Services and Mental Health Administration, of which the Bureau of Community Environmental Management is a part, contribute to this committee. All of those can bring their resources to bear on this problem.

Lastly, a few comments about the money problem. In 1972 HEW was given, after much delay, $6.5 million for lead-based paint poisoning control. Grants to local communities have been made, and local programs are underway. Because of the enormity of the housing problem, a way to effectively deal with this with limited resources did not seem feasible. A variety of different approaches and control strategies were analyzed and it was decided to focus on case finding in children.

Legal Action and Lead Poisoning

Marvin Gavin
Regional Attorney, Chicago
U. S. Department of Health Education and Welfare

As we read the awesome statistics on lead poisoning and we realize the toll it takes on the lives of thousands upon thousands of our young people each year, more and more people are beginning to look for additional ways to deal with the problem. Whereas it is important that we mobilize our medical resources to combat the deaths, the mental retardation, the lameness, the deafness and other physical injuries caused by lead poisoning, it is equally important that we call upon other disciplines in our society to combat this dread disease. It is not sufficient that we be satisfied only with making a proper medical diagnosis of lead poisoning and that we master the medical techniques of proper care and treatment of the disease. No effective fight against the ravages of lead poisoning can be made unless the legal machinery of our society is properly geared in and is made a part of our overall efforts to combat this disease.

As essential as it is that our federal, state and local legislatures and court systems play their parts in the

fight to combat lead poisoning, it is surprising that even at this late date little has been done in this area in order to combat the problem. The problem of lead poisoning has been with us for a long time. Legal action dealing with the problem is just beginning to get off the ground.

For an effective program of legal action to take place against lead poisoning, several important steps must be taken. First, the proper atmosphere must be created in our society to deal with lead poisoning. Our communities must be made aware of and educated to the problem. Secondly, out of this atmosphere there must develop some promulgation or resurrection of appropriate legislation which can control and regulate those factors which give rise to the continuation of the disease. Thirdly, enforcement of these laws by our courts and administrative bodies must take place to insure that the job is being done efficiently.

Just a few words about creating the atmosphere out of which proper legal action against the problem can be taken. It is no secret that our federal, state and local lawmaking bodies will respond only in direct proportion to the community pressures exerted on them. The problem of lead poisoning is no different. Community efforts must be made to provide a constant flow of information to the general public about the extent and nature of the problem. Education and information activities should be utilized to facilitate community awareness, understanding and support. These activities should be directed at our public officials just as much as to the social and ethnic groups most affected, parents, welfare workers, physicians, and others who are involved in this struggle. In short, I am strongly suggesting that a thoroughgoing effort be made to lobby for action in this vital area. I see this as a necessary prelude to taking effective legal action to combat the problem of lead poisoning.

It cannot be too strongly emphasized that an effective control and prevention program must be comprehensive in scope. Thus, enactment of legislation controlling all sources of lead hazards would be more appropriate in the enactments that come out of our state legislatures and municipal bodies.

Lead poisoning legislation clearly could be passed pursuant to a state or municipality's police powers or its power to protect the health, safety and welfare of its citizens. Ideally this legislation would deal with a number of items. Such an ordinance enacting a lead poisoning prevention program would:

- define its terms
- prohibit the use or application of lead-based substances in or upon exposed surfaces, fixtures, or other household objects, such as toys or furniture
- prohibit the manufacture or sale of certain articles containing lead-based substances, except in a secure container that bears a prescribed warning label
- provide for payment of blood lead determinations from public funds by qualified laboratories
- require physicians, nurses and public health officers to report diagnosed or suspected cases of lead poisoning
- prohibit the burning of lead and lead-impregnated substances, and control sale and disposal of such materials in a manner consistent with the safety and health of the public
- establish zoning restrictions to protect against lead-containing emissions in residential areas
- authorize and direct the Building Commissioner and the Health Commissioner to inspect dwelling units for lead-based substances
- provide for notice to interested and affected persons of the presence of lead-based substances in or upon exposed surfaces
- provide for the manner of safe removal of paint, putty, plaster, and other structural materials that would be consistent with accepted health standards
- prohibit eviction of families with children from dwelling units following enforcement of maintenance and occupancy standards with respect to lead and other health hazards
- provide for enforcement, penalties and appeals.

In an ideal sense, the most effective way of dealing with the problem of lead poisoning is through effective and comprehensive legislation.

In the final analysis, the most effective vehicle by which effective legal action to combat lead poisoning can be taken is our court system. In instances where there is effective and comprehensive legislation in force to control lead hazards, the job of the court system is considerably lessened and will be reduced simply to the task of enforcing a good law. If you have good legislation, the courts have an easy time enforcing that law. When you have a bad law or no law at all, the courts are put under restraint. Thus, in our present situation, however, in which we do not have comprehensive legislation in force, we place a considerable strain on our court system. Where there is no legislation or where the legislation is weak and unclear, we must go to our court system and ask it to resolve a strange and new problem within the framework of sometimes old and [illegible]iquated ways of thinking. This process is long drawn out, it is unpredictable, and many times harmful to the results we wish to obtain.

An aroused community, properly educated by health people, must demand the enactment of appropriate and

comprehensive legislation. Lawyers properly informed and sensitized to the problems must resort to the courts which can effectuate proper remedies. All these factors working in tandem must be brought about before a solution to this awesome problem can be achieved.

Political Solution

George K. Degnon
Director, Office of Government Liaison
American Academy of Pediatrics

We have been discussing the problem of lead poisoning and we have been looking at it as a medical problem; yesterday it came out that it was a housing problem; and now this morning we find through more discussion, that it is a people problem.

I would like to propose that we look even deeper into the problem of plumbism within our society and our institutions, namely into the political process. One of the functions of our domestic society and perhaps the most fundamental one, is to protect the rights of individual citizens, the most fundamental of which is the right to live. Then, you might ask what has gone wrong that in ordering our priorities we have overlooked the problem of lead poisoning? What must be done so that our concern under consideration here today might become an active issue? I think it is obvious to you that our speakers today are not going to provide you with the political solutions, but I anticipate they will give you some accounting of the political process, what has been done so far in this area, some insight as to how it has been accomplished, what yet must be done, and perhaps most importantly, how you, how we all, might participate together to achieve our objective.

It was pointed out yesterday that lead poisoning in children was identified as a problem by the Academy of Pediatrics 20 years ago. I think it is appropriate to ask why wasn't a Conference like this convened 20 years ago, or even 10 years ago, or, for that matter, 3 years ago before lead poisoning was a public issue. Public awareness was not raised to a level where those interested in the problem could anticipate achieving some success, but in these many years that have passed, some progress has been made in identifying the problem and some efforts have been made to neutralize the problem.

As I said, the Academy identified lead poisoning as a problem twenty years ago and we might ask why was it not conquered then? Well, there wasn't a great deal of public interest; there was no Congressional interest or support. Those who had identified the problem and were interested in it practically despaired of achieving success in starting a program. You might say they practically lacked hope. It is important to note that they didn't give up.

It is my hope that the speakers today will provide you some insight into the political process, how the process operates, and how your support in tandem with their efforts might help us to achieve our objectives. In further comment on the comment this morning about the people power in the political process, I think we see today a new politics in America, and I refer to it as the politics of involvement. You don't really have to look very far to see it. We saw it with the demonstrators. We see it in the environmental areas. We no longer see smoking commercials on TV. We are seeing improvements made in children's television. All of these are a result of people power, of involvement in the process.

Lead Poisoning and the Local Government

Leon M. Depres
Alderman, City of Chicago

I'd like to propose a 10-point program for local government on lead poisoning, because in combatting lead poisoning, the national government is extremely important, the state government is important, but the local government has the primary responsibility. It carries out more of the program than any of the other governments, and I think what the local government does determines what happens in the local area.

In a general way, I think there are two policies that a local government can follow. One I would call the policy of circumspection, which in my opinion perpetuates lead poisoning, and the other is the policy of eradication; and it is the policy of eradication that I want to advocate. But I want to present the policy of circumspection to you because it is really followed in some cities. To present it, I will describe an imaginary speech that an imaginary mayor might give to an imaginary conference. I want to assure you that I have no individual in mind, and that any resemblance between this imaginary speechmaker and any living person is purely coincidental. Nevertheless, this policy of circumspection has found its way into many cities, and I think it has resulted in not eradicating lead poisoning.

Now, I'd like you to imagine that this imaginary mayor has called a conference of commissioners and key personnel on health, buildings, on the data center and on public relations, and he is going to address

them on the city's lead poisoning policy, and this is the imaginary address, which I think presents the policy of circumspection:

"Ladies and Gentlemen: I have called you to discuss our city's policy on lead poisoning. Lead poisoning is a terrible thing, and our city wants to do all it can to reduce lead poisoning and hopefully some day end it.

"Lead poisoning has been with us a long time. We have done a good deal about it, we are doing a good deal about it, and we are going to continue to do a good deal about it. It is going to take time. We have to be careful and practical about what we do, because we want to help the people in our city and not hurt them, and we want a program that we can carry out and that we can afford to carry out. I don't want you to go overboard about lead poisoning. A lot of people are excited about it, but there are lots of other terrible things to take care of, too. Don't exaggerate lead poisoning and frighten people and stir up a lot of discontent."

"The first thing to do is to screen the children. That is important, because we can get federal money for screening. Screening makes our city's parents feel good if their children pass the test, and it shows we care about the children who have lead. Don't worry that we can't screen all our city's children who are exposed to lead. Just screen all you can, and keep on screening. In a few months, bring the children back and screen them again. I want our city to show the biggest possible totals of children screened."

"Now, we want enforcement of the housing code, but it has to be reasonable and workable. We can't change everything overnight. We have a big backlog of housing code cases already. Of course, you'll find a bad apple here and there, but we have to rely on the good will of our city's fine property owners or we'll get nowhere. We can't begin putting all property owners in jail. I know that some people want to impose daily fines and jail sentences in these lead poisoning cases, but let's be reasonable, and in the long run we'll accomplish more."

"Try hard to make the landlords repaint the peeling paint and plaster. You can experiment with new ideas, but just do it on a pilot basis. The pilot program always shows our city is modern and on the ball. Maybe we can get some federal money to try covering walls with canvas or plaster, but that is very costly and our city could not afford it for thousands of dwelling units. Maybe some day there will be enough for all of them, but today it just can't be done everywhere and we have to do the best we can."

"We can't make families move out of every apartment that has lead in it. Where would all those people live? They'd be looking for housing all over our city. All you'd do by moving them is stir up discontent."

"Concentrate on trying to paint surfaces, try a few pilot cases of wall coverings, and have the Health Department report all cases to the Building Department, and the Building Department report all cases to the Health Department, and that shows our city is efficient."

"We should pass ordinances, not too fast, but we should keep our ordinances up to date to show we are trying everything. As long as our city is reasonable and practical in enforcement, there is no objection to new ordinances. They show the people our city is modern and on the ball.

"Now, we can't stop families from filing lawsuits against landlords for lead poisoning that's their right, but definitely our city does not want to make its records of health available to people trying to make private profit out of these cases. Our city should be impartial and not help such cases. They just stir discontent.

"Our health records must show an improvement in lead poisoning. People want improvement. If we go ahead with our program, we are bound to show improvement and the figures will show it. Any cases of lead poisoning you find in the screening program should be referred only to the city agencies for treatment. We know more about it and can do a better job. Don't go referring cases to private hospitals. Lord knows what those doctors will say or do or find. With all this hysteria about lead poisoning, if you encourage private doctors to handle these cases, they'll begin calling every headache a case of lead poisoning. They'll begin saying a lot of well children have high lead levels and they'll begin encouraging a wild housing enforcement program and stir up discontent. Keep the cases where we know how to handle them – in the city agencies."

"Watch the statistics carefully. I know they will show some improvement unless they are misleading. Our city does not want misleading statistics, and you have to interpret statistics properly – you know how to do that. Under our city's program, the figures should show that lead poisoning is going down. Maybe you can increase the age of the children you screen. We want only accurate statistics. And watch those deaths. A lot of overenthusiastic doctors, and some subversive ones, will be calling everything death from lead poisoning. Don't let that happen."

"We know our program will reduce lead poisoning deaths and the figures should show it. If they don't, they'll stir up a lot of discontent. Nothing is perfect, but I want you to be sure to let people know where the blame lies. It does not lie in our city. Remind them that the Federal Government is holding back the money, and the state government, too."

"Remind them that lead poisoning is a matter of education. If a mother looks after her child properly, there is no reason for lead poisoning. I think we can get a grant to produce a film on how to care for a child properly and prevent lead poisoning, and then people will see that it isn't all just a problem of enforcement, but the mothers are too often to blame. Your mother and my mother didn't let their children get lead poisoning, and there was lead paint in our homes."

"Remind them that the paint dealers are to blame, too, for not labeling the paint properly. And be careful not to let subversive groups use lead poisoning as an excuse to organize. Deal with citizens individually."

"Now, I want our city to get all the federal and state money we can, for screening, for new hardware, for new tests. That shows our city is up to date and on the ball. The whole story of what our city is doing has to be told to the people. They should know how many children are screened; that story can't be told too often. All pilot programs and new machines and new tests and screening programs should be told to the people so they see what our city is doing."

"Now, once in a while the press will attack you. They'll be very unfair. All they care about is sensation and lies and selling papers and hurting the program. Never give them any information. All information must come through the public relations office. Do not talk to the press. But if the press attacks come and there is sensationalism about a child dying or enforcement or the statistics, remember that our city's administration will stand behind you. Ride with the punch, because every unfair press attack comes to an end and is forgotten, but we wouldn't want to find that any of you leaked anything to the press."

That is the end of the imaginary mayor's talk. But the policy which I would describe is a policy of circumspection, and I think that policy of circumspection leads to the perpetuation, perhaps even to the increase of lead poisoning.

And I would like to give you now what I think is a practical ten-point program for local governments.

Point one, which is the most important of all, is that the city must have determined to end lead poisoning. That is the basic point and that is the most difficult.

Second, the city government must pass modern, effective legislation. Legislation alone, without determination and without enforcement, is not sufficient. Even without modern, up-to-date ordinances, an effective program cannot be founded.

Third, enforce the legislation. Do it speedily, innovatively, and firmly. We have to find new ways of enforcing legislation, ways of doing it effectively, and then doing it firmly. I think that fines and imprisonment are very, very important in making the legislation effective. And remember, too, that although housing is the most important kind of legislation to be enforcing, air pollution is also important. They are finding now that lead poisoning comes also from air pollution.

Fourth, the local government should see that public health treatment of lead poisoning is done on an epidemic basis as to both prevention and treatment. That is, the Health Department has to go after lead poisoning with the same zeal in prevention and treatment that it goes after other epidemics. Dr. Dubos called lead poisoning the silent epidemic, but it is an epidemic and has to be treated that way.

Fifth, I think the city has to adopt a policy of the most extensive possible deleading and sheathing of the affected dwelling units.

Sixth, because the deleading and sheathing won't occur immediately and overnight, there should be interdiction of dangerous housing to vulnerable children. The housing should be labeled dangerous, should be posted as dangerous, no rents should be payable on it, and either immediately or very quickly it should be prohibited for occupancy by young children.

Seventh, the one policy of local government should be to obtain maximum federal and state aid. There is federal and state aid available, and that has to be obtained to the maximum possible amount.

Eighth, part of the local government policy should be to stimulate federal policy on lead poisoning.

Ninth, full disclosure of facts, full disclosure of everything about lead poisoning should be made public so that the statistics are accurate, the locations are given and the information is available to all.

Tenth, education, especially the kind of education that encourages local community groups, tenant unions, any form of organization, especially of the victims of lead poisoning, and those who identify with the victims of lead poisoning should be an integral part of our program.

Welcoming Remarks — Wilmington

Harry G. Haskell
Mayor of Wilmington

Mayor Haskell welcomed the delegates and spoke on the legislative problems associated with lead poisoning and related problems of the inner cities. He estimated that 65% of Wilmington children are in some way disadvantaged. He stressed that lead poisoning is but *one* of many avoidable ghetto diseases which may hinder ghetto children's intellectual development rather than an inherent inferiority.

An Overview

Sidney J. Sussman, M.D.
Temple University

Dr. Sussman described lead poisoning as a disease of inner city children more than other children. Today more of the inner city children have asymptomatic blood lead elevations than the symptomatic variety as in the past.

He emphasized the point that one of the major methods of combating this problem is in the political arena in which physicians have previously shown reluctance to become involved. As an example he pointed out that the success in getting a Philadelphia lead screening program started is attributed to the formation of "The Coalition Against Lead Intoxication," consisting of community members and professionals.

Screening for Lead Poisoning

Elizabeth M. Craven, M.D.
Wilmington Medical Center

Dr. Craven pointed out that with increased screening, there was an increase in the symptomatic cases. Although lead poisoning may account for only 3% of all poisonings in children, it may account for as much as 10% of the mortality.

Sources of lead and problems in screening technology were discussed and the need for standardizing of methods stressed. Dr. Craven assented that blood lead appears to be the most feasible method for screening at the moment and micro blood techniques (Delves cup, carbon rod, anodic stripping voltammetry) are being field tested.

The New York Experience

Vincent Guinee, M.D.
New York City Health Department

During two years of intensive lead screening activity, 1970-71, in New York City over 200,000 tests were performed and 4,574 children were identified with blood lead levels of 60 micrograms or higher. Repairs in 3,500 homes resulted. One out of six was repaired by the landlord, the remainder by the city emergency repair program.

Dr. Guinee estimated there were still some 5,000 children in New York City with levels of 60 micrograms or above that have not been screened. An estimated 450,000 apartment units with 120,000 children living in them constitute the lead poisoning hazard in New York City.

When apartments that have children with elevated blood levels are inspected, about 80% have some high lead content paint identified. A lead poisoning "case" in New York City is currently defined as a child with 60 micrograms or more on a single determination.

When a child is found to have a blood lead level of 60 micrograms, the Health Department notifies the physician submitting the specimen, and a nurse and sanitarian visit the home and plan for medical supervision and attempt to determine the source of lead available to the child. If the paint tested contains more than 1% lead, the owner is ordered to correct the condition within 5 days; if he fails to comply, the work is done by the city's emergency repair program.

An increase in the amount of cases detected is a result of the intensity of the screening effort. While more are found in summer months, a significant number are detected throughout the year.

Among 5,000 black children tested there were 9% showing 60 microgram levels, and among 5,000 Puerto Rican children there were only 3% showing the same level.

In addition to finding cases and repairing buildings, they have been doing operational research and epidemiology research that is important not only to guide programs, but also to focus attention on areas that need to have special attention.

Despite the New York City Health code of 1959 banning the interior use of lead paint above 1%, a store sampling survey showed 25 of 76 manufacturers did not comply. Paints labeled "safe for cribs" contained as much as 16% lead.

The Wilmington Experience

Barbara B. Rose, M.D.
Newcastle County Health Department

Of the 42 cases of lead poisoning reported in Delaware since 1961, all but one have been reported since July 1969 (when it became a reportable disease). The 39 cases reported from the city of Wilmington occurred in children under age 6 and who live in houses in the inner city of Wilmington.

Follow-up of 23 hospitalized cases from the Wilmington disease center (1961-1971) showed: 2 deaths; 5 children moderately to severely retarded who will probably require custodial care; 3 children in special education classes; and the remaining 13 children with no gross neurological defects in regular classes.

Two survey results were reported: one in a hospital outpatient department and the other in day care centers and a well-child clinic. Both small surveys (totalling 171 children below age 6) showed that 34% of those tested had blood lead levels above 40 micrograms/100 ml whole blood. A total of 8 children required hospitalization and treatment (5%).

As a result of the surveys a housing follow-up protocol was established for homes involved in lead poisoning cases. To date 21 have been corrected by either rehabilitation or moving the family, and another 14 are pending. Application for a city-wide screening program is being made. (Editor note: Wilmington now has a Federally-funded program).

The Federal Role in Preventing Lead-Based Paint Poisoning

U.S. Senator William V. Roth, Jr.

The toll which this disease takes is especially distressing because here, like in other areas, we know basically what causes it and what needs to be done to remove it. Although lead paint poisoning may not present as many medical mysteries to us as other diseases, its consequence is a social problem of tremendous magnitude. Great legislative and administrative ingenuity will be needed to mount a successful attack by different levels of government.

As co-sponsor of the legislation before the Senate, S3080 which would renew and strengthen this particular statute, I am convinced that Federal action is required in this area of childhood lead paint poisoning for several reasons: the Federal Government's power is really more equal to the task of preventing lead paint poisoning than is other units of Government; constitutional authority to regulate interstate commerce is necessary to the setting of standards for lead content in paint; the national government's huge role in housing allows it to get at this problem; the Federal Government possesses an advantage in providing comprehensive coordination to the efforts of different Government levels and in publicizing the causes, symptoms, and remedies of lead paint poisoning. I am not arguing for exclusive Federal jurisdiction in this matter, only a Federal role in it.

Bill S3080 would fund more adequately, community programs to detect and treat the cause of lead paint poisoning. Early detection is outranked in importance only by prevention itself. It would provide for State and local programs to identify areas where the risk of poisoning is high and to eliminate the heavily leaded paint surfaces. A third part of an adequate Federal program on this crippler would be the adequate funding of research into the nature of extent and means of removing lead paint as a threat. Tied to this is a very important need to provide more and better information about it to parents, doctors, other health people, and of course the public at large. Further initiative to combat lead poisoning in the future would be to provide for the practical elimination of lead from paint. I feel that regulatory responsibilities are also called for in the housing area. HUD could appropriately move to eliminate old lead-based paint from walls and public houses, HUD-owned properties and in its rehabilitation work, at the local level and building code laws. Adequate funding of the Lead-Based Paint Poisoning Elimination Act would be a major step in the right direction.

Model Legislation at the State Level

Senator Louise T. Conner
Delaware State Senate

The Delaware Senate is working on Lead Poisoning Legislation based somewhat upon, but more limited in scope, than the Massachusetts Bill that is now law. The Massachusetts Bill applies to lead in paint and pottery and creates a Director of Lead Poisoning Control within their Department of Public Health. That Bill makes lead poisoning a reportable disease and authorized the initiation of educational and mass screening programs as well as area inspection programs to locate lead belts. The Senator stressed the need for interested citizens to write to other Senators to support both legislation and funding.

The Need for Combined Legal and Medical Responsibilities In Childhood Lead Poisoning

Jonathan Stein
Attorney, Welfare Rights Organization
Commission of Legal Services, Philadelphia

The main emphasis in eliminating lead poisoning must be on legislation and effective programs. Much of the technology and etiology of the problem is known but there has been a conspicuous failure on the part of the medical and legal professions in their resolve to take responsibility for its correction.

An effective program must have a bilateral approach in coupling effective screening of children with inspection and correction of housing. Only through local community action and pressure would there be an

effective voice in Washington to direct the Federal Government to get involved in the housing aspect of the problem and to increase the amount of money allocated for the entire country's lead poisoning efforts (7.5 million dollars).

Mr. Stein described some of the activities of the lead poisoning coalition in Philadelphia that helped in increasing both the size and commitment of the lead poisoning program.

Problems Relating to Enforcement of the Housing Code

Mr. Timothy Jackson
Commissioner of Licenses and Inspections
City of Wilmington

Mr. Jackson's main point was the financial costs involved in the enforcement of the housing code and elimination of the lead paint from the houses where it was detected. He estimated that it would cost between 600 and 1500 dollars per house to remove the lead-based paint, either by burning or scraping. He noted that X-ray fluorescent analyzers can help in detection of lead on walls but also cost a lot of money in initial investment. He stressed the need for Federal funds to be used for paint removal as well as screening and treatment.

Lead Industries' Role in Preventing Lead Poisoning in Children

Jerome F. Cole, Sc.D.
Lead Industries Association, Inc.

Dr. Cole summarized his organization's past role and continuing efforts aimed at reduction of childhood lead poisoning from old lead paint and improperly glazed pottery. He supported the further reduction (below 1%) of lead content in interior paints to levels consistent with the technical ability of the paint industry to produce quality products.

He enumerated "Seven Steps to Prevention" which included:

1. Alert and warn parents and others who live in dwellings which have leaded paint in interiors.
2. Remove sources of lead that children can eat.
3. Take steps to keep any child suspected of eating lead from further exposure.
4. Physicians, Public Health Nurses and others should watch for early, vague, non-specific symptoms of lead absorption.
5. Quick and accurate diagnosis prevents serious consequences.
6. Proper and careful treatment should start immediately after diagnosis of lead intoxication.
7. When a case is found, check other children in the home immediately for possible signs of lead absorption.

The Problem of Lead in Paint

R. W. Laurell
E.I. duPont de Nemours and Co.

Mr. Laurell emphasized that he was not speaking for the entire paint industry but only as a representative of the duPont finishes Division. He felt that paint presently produced is safe but that old paint used prior to the 1950s was hazardous to children because of both lead content (up to 50% on interior surfaces) in each layer and continued build-up from successive paintings.

DuPont, acting independently, recommended in 1940 that lead content be no greater than 0.1% in children's furniture and toys. By 1970 a standard of 0.05% was adopted in both interior and exterior latex paints (with the exception of a water based primer). The proposed FDA 0.06% standard can be met as long as occasional raw material contamination is allowed for.

Control of Sources of Lead from Food and From the Atmosphere

Lloyd Tepper, M.D., Sc.D.
University of Cincinnati

Dr. Tepper mentioned that in addition to pottery and glazing, lead contamination could occur in illegally prepared whiskey. His laboratory has also recently become interested in lead in food.

Twenty women volunteers from eight communities across the country collected their total urine and fecal output for 10 days. In this study the average daily lead ingested per woman was determined to be between 90 and 150 micrograms, an amount lower than previously reported.

Except in certain industrial situations, the transcutaneous absorption of lead is not worth considering. However, ambient air lead levels in urban, suburban and rural areas do deserve closer scrutiny. The recent situation in El Paso, Texas, illustrates how airborne lead from industrial

emissions can be a problem. In an area called Smeltertown, the children of a Mexican-American community were subjected to the risk of lead poisoning from an operating lead smelter. The blood lead levels in the children of the approximately 100 families there ranged between 40-80 micrograms. A few were above 80 micrograms but all were asymptomatic. The adults were all normal. The presumption was that the children get their lead from the fallout in the dirt which exceded 1% lead.

Dr. Tepper had sampled the lead content of the air in twenty locations in eight communities across the country, varying in population, vehicular density and in type of industries nearby. Atmospheric lead levels varied from a low of 0.17 micrograms per cubic meter in Los Alamos, New Mexico, to levels above 3 micrograms per cubic meter in Pasadena, California. Despite this variation in ambient lead, the blood lead levels of adults in all these areas were remarkably uniform. In almost every instance, smokers had blood lead levels two micrograms percent higher than non-smokers.

The Community Approach To Lead Poisoning

Mrs. Veronica Singleton
Welfare Rights Organization
Philadelphia

Mrs. Singleton felt that out of pending Federal Legislation, some money will come but more money and more kinds of Legislation are needed to substantially change the danger of lead poisoning. Federal agencies should have stricter standards about lead content of painted walls at the time of sale or rental of housing. City governments that take over the titles of dilapidated housing of people unable to keep up mortgage payments, could eliminate lead in those dwellings by renovations. She calls the health department whenever she learns of a case of lead poisoning and tells the mother to notify the health department if she is moving, so that their dwelling may be posted as unfit in order to avoid continued exposure of other children. Local welfare rights groups need to work harder to get city governments to develop detection programs, stricter housing codes and to enforce existing ordinances.

A child's life, eyesight and brain are far more precious than any expense to industry in removing the lead from new paint which will only add to the older layers of high lead paint. Lead poisoning is a man-made disease that has been perpetrated by local realtors and owners of dilapidated housing.

She described her testimony before Senator Edward M. Kennedy's subcommittee hearings on lead poisoning and stressed that face to face contact is the only means of truly informing the people.

Development of a Program for Lead Poisoning Control

Mary Lou Anderson
Health Planner
Model Cities Program
Wilmington, Delaware

Mrs. Anderson expressed unhappiness over the fact that at the present time there was no lead control program in Wilmington. Almost 100% of all the housing and model cities' areas are dangerously lead-contaminated. The only option is to condemn buildings, but then what? Inertia on the part of the medical profession, State legislators, State and local health departments, has made it hard to get basic legislation, screening programs and public information measures started. Until these problems are solved, there is a feeling of futility about ever solving the lead poisoning situation.

Developing Community Action

James Baker
Wilmington Leadership Training Program

Mr. Baker expressed a reluctance to tell physicians what to do but felt that in the problem of lead poisoning they should do much more to translate their theories into community action. Community action is not synonymous with participating in conferences and the medical profession should become more involved in the political process. The issues of social change implicit in solving lead poisoning must not be avoided.

Putting It All Together

Robert E. Novick, Director
Bureau of Community Environmental Management
Health Services and Mental Health Administration, H.E.W.

Mr. Novick expressed the feeling that the central theme that ran through the Conference was that the poisoning of children with lead-based paint is a community problem. It is a problem that has not yet been recognized for its' full social impact by the

Government agencies, Congress, State legislatures and city councils.

In dividing lead poisoning into three areas – medical, environment and social – the medical area has made the most progress. Social progress has only progressed as far as community action groups but it is in the environmental area that results are so discouraging. Despite the presence of 30-40 million potentially hazardous dwelling units, including some 6-7 million dilapidated units, we still use the archaic methods of blow torches and putty knives to remove the lead-based paint from the walls and window sills.

A serious concern pointed out by the group has been the lack of commitment within those organizations, agencies, groups and industries that control and manage the housing stock. One cannot separate out completely lead poisoning from the many other health and housing problems that affect the inner cities but an attempt to focus some resources on one aspect of a problem may result in solving some of the other parts.

Mr. Novick pointed out that just two years prior to the Conference there was no Federal Legislation and no identifiable resources within the health establishment of the Federal Government of any significance. Much of the identifiable progress recently has been made by voluntary community effort and by the use of local resources without much Federal support.

Mr. Novick said: "Incidentally, I am very firmly on record endorsing that .06 standard. I think it's overdue. I think it's feasible and accomplishable. We also talked about lead in food. We talked, or someone mentioned burning old battery cases, the El Paso smelter was mentioned, air pollution with lead. I think to a certain extent these things are diversions. The fact is that there are somewhere between 30 and 40 million dwelling units in the United States with lead-based paints on the walls today. This is the primary cause of the problem and should be the primary thrust of all of our activities. Let's not get lost chasing paper tigers."

EDITOR'S ADDENDUM

A review of the Wilmington presentations and the meeting's effect on the participants reveals its true impact.

A small city, Wilmington, discovered a small "epidemic" of lead poisoning within its walls. The fractured and fragmented methodology by which a small scale proposed control program evolved became an object lesson for most small to medium sized communities.

The intensity of interest in the need for organized community action and the need for community organizers dominated all phases of the conference. The most eloquent expression of the problems of childhood lead poisoning came from community groups themselves. The lack of acceptance of more responsibility on the part of medical, legal and political elements in our society was brought out in the open.

Throughout the conference's discussions, the lack of awareness of the dimensions of the problem on the part of the public, the Government and the media, was emphasized, as well as the importance of single individuals combating established institutions through community political activism.

Above all, the conference emphasized the need for a continued Federal presence in both the provision of seed money and technical expertise.

Roger S. Challop, M.D.
February 1973

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